

Sleep disordered breathing- An overview

BC Muddugangadhar¹, G S Amarnath², Dikshit Suchismita³, Tripathi Siddhi⁴

ABOUT THE AUTHORS

1. Dr. Byrasandra Chennappa
Muddugangadhar,

ASSISTANT PROFESSOR,
DEPARTMENT OF PROSTHODONTICS
INCLUDING CROWN AND BRIDGE AND
IMPLANTOLOGY
M R AMBEDKAR DENTAL COLLEGE &
HOSPITAL, BANGALORE, KARNATAKA,
INDIA.

2. Dr. G S Amarnath

PROFESSOR AND HEAD
DEPARTMENT OF PROSTHODONTICS
INCLUDING CROWN AND BRIDGE AND
IMPLANTOLOGY
M R AMBEDKAR DENTAL COLLEGE AND
HOSPITAL, BANGALORE, KARNATAKA,
INDIA

3. Dr. Dikshit Suchismita
POST GRADUATE STUDENT

DEPARTMENT OF PROSTHODONTICS
INCLUDING CROWN AND BRIDGE AND
IMPLANTOLOGY
M R AMBEDKAR DENTAL COLLEGE &
HOSPITAL,
BANGALORE, KARNATAKA, INDIA.

4. Dr. Tripathi Siddhi

DEPARTMENT OF PROSTHODONTICS
INCLUDING CROWN AND BRIDGE AND
IMPLANTOLOGY
M R AMBEDKAR DENTAL COLLEGE &
HOSPITAL,
BANGALORE, KARNATAKA, INDIA.

Corresponding Author:

Dr. B C Muddugangadhar,
E-mail: drmuddu@yahoo.com

Abstract

Sleep disordered breathing has been used to describe a spectrum conditions ranging from snoring as a mild form to Obstructive Sleep apnea (OSA) which is a more severe form. Upper airway resistance syndrome falls in between the two. Oral appliance therapy and therefore dentistry has become an important component of treating patients with upper airway sleep disorders. This article places oral appliances as a treatment of choice for snoring and mild to moderate Obstructive sleep Apnea in certain patients and ahead of surgical procedures for all but the most severe patients.

Key words- Obstructive sleep Apnea, Oral appliances, Upper airway resistance syndrome

Introduction

Sleep disordered breathing has been used to describe a spectrum conditions ranging from snoring as a mild form to Obstructive Sleep apnea which is a more severe form. Upper airway resistance syndrome falls in between the two. These disorders are commonly the result of conditions or diseases which cause partial or complete obstruction of airway when the patient is supine.

One of the first references of a patient with medical and physical characteristic features of sleep disordered breathing was made in a famous novel by Charles Dickens known as the Pickwickian papers where he described a character named Sam.¹ The condition was named as the Pickwickian syndrome. In 1956 Burwell referred Pickwick Papers and its obese character when describing the Pickwickian syndrome as consisting of obesity, hypersomnolence, hypoventilation, and cor pulmonale.² In 1960 this disease was named as Obesity Hypoventilation syndrome. Over the years further research in this field have resulted in formation of a new medical specialty known as the "sleep medicine".

There are several modalities of treatment for obstructive sleep apnea. The use of oral appliances for management of airway obstruction dates back to early 1900 when the French stomatologist Pierre Robin used oral appliances to manage upper airway obstruction of neonates born with Pierre Robin syndrome.³ Since then Oral appliance therapy and therefore dentistry has become an important component of treating patients with upper airway sleep disorders. In 1995 the American Sleep Disorders Association published its parameters of Care document that recommends treatment modalities for Upper airway sleep disorders (UASD) where oral appliances are a treatment of choice for snoring and mild to moderate OSA in certain patients and ahead of surgical procedures for all but the most severe patients.⁴

Etiology

Obstructive sleep apnea has complex etiology and consists of multifactorial interplay between the neuromuscular systems, airway and anatomical structures.⁵ The upper airway is basically a soft tissue tube, the patency of which is maintained in part by the activity of muscular groups of which the tensor veli and genioglossus muscles are highly important members. The action of these muscle groups has an important influence on the physiology of sleep disorders. The airway of a patient with snoring is obstructed due to tongue or hyoid bone and overlying soft tissues dropping back towards the posterior wall of pharynx when the patient is supine in order to get sufficient oxygen to the lungs. There is increase in velocity of air passing through narrow airway which in turn causes vibration of soft tissues especially the soft palate and uvula. This vibration is the sound of snoring.⁶⁻⁹ Obstructive sleep apnea patients may suffer from hundreds of apneic events per night, which by definition are blockages of airway lasting more than 10 seconds.¹⁰ There are several factors which predispose to obstructive sleep apnea, the most important being obesity. Clinic based and epidemiological studies demonstrate a strong association between obesity and obstructive sleep apnea. Potential mechanisms to be considered include: (1) alterations in upper airway structure; (2) alterations in upper airway function; (3) alterations in the balance between ventilatory drive and load and (4) obesity induced hypoxemia.¹¹ Other factors include head posture, inflammation of surrounding soft tissue, tonsils, adenoids¹²⁻¹³ or epiglottis, tumor, structural compromise, retrognathia¹⁴⁻¹⁷, paralysis of intraoral muscles, nasal septal deviation, longer anterior facial height¹⁸ etc.

Clinical characteristics

- Overweight with a large neck¹¹
- Hypertensive¹⁹
- Does not get adequate amount of sleep
- Dozes during the day
- Accident prone
- May awaken from sleep gasping for air
- Suffers from reflux
- Often is retrognathic¹⁴

Diagnosis

Diagnosis is based on medical history, physical examination and testing. Polysomnogram or sleep study is the gold standard. This study records sleep staging like electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), and physiological variables like sleep positioning, respiratory activity, oxygen saturations, blood pressure and Ecardiogram. Polysomnography reveals the quality and quantity of sleep. Polysomnogram (PSG) provides apnea index (AI), Hypoapnea index (HI) and Apnea-Hypoapnea index (AHI). Polysomnography reveals that OSA patients will characteristically fall asleep, experience an upper airway

blockage, exhibit loud snoring, stop breathing, experience a drop in blood oxygen levels, attempts to breathe, arouse, gasp for air, breathe for a few seconds and fall back asleep. Once the SaO₂ (saturated oxygen) drops to a certain level, patients will have an arousal and begin breathing again.¹⁹

Treatment modalities

Non specific therapy

Behavior modification include changing the sleep position from the supine position to the side position; this can be accomplished by placing a tennis ball in the centre of the back of their dress or by positioning a pillow such that they cannot roll on to their back (positional training). The avoidance of alcohol and sedatives; they may also act as muscle relaxants, reducing airway patency. In obese patients, weight loss should be recommended.¹⁹ Even a 10% weight loss can reduce the number of apneic events for most patients.

Specific therapy

1. Oxygen administration²⁰

Oxygen is sometimes used in patients with central apnea caused by heart failure. Oxygen at the correct flow rate when used in conjunction with nasal continuous positive airway pressure (CPAP), however, in many cases corrects this problem.

2. Pharmacological agent

Thyroid hormone supplementation might lead to significant correction of the apnea if this is the sole problem. Control of blood sugar levels has a moderate effect in controlling the diagnosed obstructive sleep apnea. Certain medications which increase respiratory drive help some patients.

3. Continuous positive airway pressure appliance (CPAP)

Continuous positive airway pressure is the gold standard in treatment. CPAP treats patient by pumping room air under pressure through a sealed face mask or a nose mask through the upper airway to lung,²¹ however the treatment is associated with poor patient compliance due to lack of portability, pump noise, dryness of the airway passage, and mask discomfort.²²⁻²⁴

CPAP is administered at bedtime through a nasal or facial mask held in place by velcro straps around the patient's head. The mask is connected by a tube to a small air compressor. The CPAP machine sends air under pressure through the tube into the mask, where it imparts positive pressure to the upper airway. This essentially "splints" the upper airway open and keeps it from collapsing in the deeper stages of Rapid eye movement sleep. The pressure acts much in the same way as a splint, holding the airway open.

4. Surgical correction

Uvulopalatopharyngoplasty, partial tongue resection

tracheostomy, lingual plasty, genioglossal advancement with hyoid myotomy and suspension nasal septal surgery hyoid bone suspension, and mandibular advancement osteotomy are the various surgical modalities suggested for obstructive sleep apnea.¹⁹

In nasal, septal and adenoid surgery weak or malpositioned cartilages around the nostrils, droopy nasal tip or excessively narrow nostrils, nasal septal deviation and enlarged adenoids are all indicated for surgical interventions.

Tonsillectomy allows the removal of redundant tissue and hence increases the caliber of the throat thereby reducing blockage to breathing. Genioglossus tongue advancement procedure produces a larger space between the back of the tongue and the throat thereby creating a wider airway. Uvulopalatopharyngoplasty (UPPP) involves the removal of part of the soft palate, uvula and redundant peripharyngeal tissues, sometimes including the tonsils. Laser-assisted uvulopalatoplasty like UPPP may decrease or eliminate snoring but not eliminate sleep apnea itself. Maxillomandibular advancement or double jaw advancement is a procedure where the upper and lower jaws are surgically moved forward. Radiofrequency tissue volume reduction (RFTVR) is a surgical method which uses radiofrequency heating to create targeted coagulative submucosal lesions resulting in shrinkage of the inner tissues leaving the outer tissues intact.

Hyoid suspension is a procedure which was developed specifically for the treatment of OSA. The operation advances the tongue base and epiglottis forward, thereby opening the breathing passage at this level.

In Tracheostomy any area of blockage to breathing, from the nose to the voice-box, is bypassed by a hole placed into the windpipe.

5. Oral appliance therapy

They can be divided into three general groups: soft palate lifters (SPL), tongue retaining devices (TRD), and mandibular advancement appliances (MAA). The first category is virtually no longer in use today. The second category is used very seldom, mainly if there are dental reasons precluding the construction of MAA. The last category (MAA) is by far the most common type of dental appliance in use today. It protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep.²⁵⁻²⁹

Mechanism of action

Oral appliances are worn only during sleep and work to enlarge the airway by moving the tongue or the mandible forward.

Indications of oral appliance therapy

Given by American Academy of sleep medicine:² (i) Oral appliances are indicated for use in patients with primary

snoring or mild OSA who do not respond or are not appropriate candidates for treatment with behavioral measures such as weight loss or change in sleep position; (ii) Patients with moderate to severe OSA should have an initial trial of nasal CPAP because greater effectiveness has been shown with this intervention than with the use of oral appliances; (iii) Oral appliances are indicated for patients with moderate to severe OSA who are intolerant of, or refuse treatment with, nasal CPAP. Oral appliances are also indicated for patients who refuse or who are not candidates for tonsillectomy and adenoidectomy, cranial facial operations or tracheostomy.

Contraindications

Severe periodontal disease, Existing TMJ disorders, Painful masseter muscles, Incomplete dentition which compromises retention of the appliance, atrophic edentulous ridges as evidenced by poor denture retention, severe hypoxemia, severe OSA, growing children, protrusive range of mandible < 7 mm, mouth opening restricted to 30 mm or less, unmotivated patients, central sleep apnea.

Mandibular advancement devices

As mentioned earlier the first use of mandibular advancement devices was suggested by Pierre Robin in 1903.³ It protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep. These devices can be either fixed (i.e., the protrusion distance cannot be changed), or variable (i.e., protrusion can be increased or decreased). The final protrusion distance represents a delicate balance between side effects and efficacy. It has been shown by various studies that mandibular advancement devices significantly reduce AHI³⁰⁻³⁴.

Currently available appliances¹⁴

First generation

They are one piece appliance with no ability to advance the mandible incrementally.

Second generation

These appliances are principally two piece in design and offer the potential for incremental advancement.

Third generation

They not only permit incremental advancement, but also lateral movement of mandible.

Fabrication technique

The mandibular advancement devices require good retention, sufficient protrusion to maintain airway, minimal vertical opening and full occlusal coverage. With either fixed or adjustable device, the initial position of mandible is generally 70 to 75%. The interocclusal records are made with patient protruded to 70 to 75% of

the maximum protrusive movement and the device is fabricated. Single position stock devices (Therasnore) are fabricated in the form of two rigid plastic shells in the shape of impression trays joined into a single unit. Retention is obtained from the thermoplastic fill material. Adjustable mandibular advancement devices (Quinsight, P M positioner) are plastic impression trays filled with thermoplastic material for fitting to the patient and a mechanism for adjusting the mandible.

Titration of oral appliances

It is a method of slowly moving the mandible anteriorly or posteriorly using adjustable mechanisms until symptoms are relieved with minimal possible protrusive position. Recall is necessary at 2 weeks, 1 month and every 6 months interval.

Various studies have proven that mandibular advancement devices are an effective solution for significant number of patients with mild to moderate obstructive sleep apnea.³⁵⁻³⁶

Tongue retaining devices

Cartwright and Samelson described the tongue retaining device in 1982³⁷. They are excellent devices for edentulous patients and patients with temporomandibular joint sensitivity. It is a one piece device made of non rigid vinyl material without thermoplastic material to adapt to teeth. It consists of Kelgauge template of various sizes. Patient is asked to protrude the tongue into the hollow bulb template of the appropriate size and interocclusal records are made. Appliance is fabricated with the help of these records. There are several advantages of Tongue retaining devices. They do not require retention from dentition, minimal adjustments are required and cause minimal sensitivity to teeth and temporomandibular joint.

Side effects of oral appliance

Patients may encounter any of the following: difficulty in going to sleep, salivation, chapped lips, dry throat, sore teeth, sensitive temporomandibular joints, and posterior teeth not occluding properly upon awakening.³⁸⁻³⁹ Difficulty to sleep can be resolved by suggesting that the patient go to sleep without the oral appliance; when his or her snoring becomes prominent to the patient's bed partner, the partner should awaken patient to insert the oral appliance. Sore teeth and sensitive temporomandibular joints should disappear within a couple of hours after the oral appliance is removed in the morning. If symptoms persist, the oral appliance requires immediate removal and possible adjustment by the clinician.

Summary

Oral appliances may increase upper airway size at multiple levels and this may be important in producing

their clinical effect. The literature describing oral appliance therapy for OSA has improved dramatically in the last few years in terms of both quantity and quality. The role of these appliances in adolescents and children has yet to be evaluated in a comprehensive or systematic way. Studies addressing these issues will advance the field of oral appliance therapy and improve the care being delivered to patients with OSA.

References

1. C. Dickens. Posthumous Papers of Pickwick
2. Waldhorn RE. Sleep Apnea Syndrome. Am Fam Physician.1985; 32:149-166.
3. Robin P. Glossoptosis due to atresia and hypertrophy of the mandible. Am J Dis Child. 1934; 48: 541-547.
4. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. American Sleep Disorders Association. Sleep 1995; 18: 511-3.
5. Mimi yow. An overview of oral appliances and managing the airway in obstructive sleep apnea. Semin orthod 2009; 15: 88-93.
6. Schwab RJ, Gupta KB, Geftter WB, Metzger LJ, Hoffman EA, Pack AI. Upper airway soft tissue anatomy in normal and patients with sleep disordered breathing: Significance of the lateral pharyngeal walls. Am J Respir Crit Care Med 1995; 152: 1673-89.
7. Brown LK. Sleep apnea syndromes overview and diagnostic approach: Mt. Sinai J Med 1994; 61: 99-112.
8. Chaudhary BA, Smith JK. Obstructive sleep apnea syndrome. J Med Assoc Ga 1991; 80: 541-545.
9. Goodday RH. Nasal respiration, nasal airway resistance, and obstructive sleep apnea syndrome. Oral Maxillofac Surg Clin North Am 1997; 9: 167-77.
10. Remmers JE, DeGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. J Appl Physiol 1978; 44: 931-8.
11. Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. Sleep 1996; 19 :104-15.
12. Shintani T, Aaskura K, Kataura A. The effect of adenoidectomy in children With OSA. Int J Pediatr Otolaryngol 1998; 44: 51-58.
13. Chan J, Edman JC, Koltai PJ. Obstructive sleep apnea in children. Am Fam Phys 2004; 69: 1147-1154.
14. Johal A. A review of the use of mandibular advancement appliances in sleep disordered Breathing. Dent Update 2008; 35:230-235.
15. Bacon WH, Kreiger J, Turlot JC, Stierle JL. Craniofacial characteristics in patients with obstructive sleep apnea syndrome. Cleft Palate J 1988; 25:374-8.
16. Imes NK, Orr WC, Smith RO, Rogers RM. Retrognathia and sleep. JAMA 1977;237:1596-7.
17. Jamieson A, Guilleminault C, Partinen M, Quera-Salva MA. Obstructive sleep apnea patients have craniomandibular abnormalities. Sleep 1986; 9:469-77.
18. Torrne LPM. The long face syndrome and the impairment of nasopharyngeal airway. Angle Orthod 1990; 60: 167-176.

19. Ivanhoe JR, Attanasio R . Sleep disorders and oral devices. *Dent Clin North Am*. 2001; 45:733–758.
20. C Sunitha, S Aravind Kumar. Obstructive sleep apnea and its management. *Indian J Dent Res*. 2010 ; 21:119-124.
21. Ferguson KA, Ono T, Lowe AA, Keenan SP, Fleetham JA. A randomized crossover study of an oral appliance versus nasal continuous positive airway pressure in the treatment of mild-moderate sleep apnea. *Chest* 1996;109: 1269-75.
22. Dowe R III, Perkin RM, MacQuarrie J. Nasally applied continous positive airway pressure use in children with obstructive sleep apnea younger than 2 yrs of age. *Chest* 2000;117:1608-1612.
23. Hoffstein V, Viner S, Mateika S, Conway J. Treatment of obstructive sleep apnea with nasal continuous positive airway pressure. Patient compliance, perception of benefits and side effect. *Am Rev Respir Dis* 1992;145: 841-5.
24. Kribbs NB, Pack AI, Kline LR, Smith PL, Schwartz AR, Schubert NM, et al. Objective measurements of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993;147:887-95.
25. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. *Sleep Breath* 2007; 11:1–22.
26. Ivanhoe JR. Treatment of upper airway sleep disorder patients with dental devices. *Clinical Maxillofacial Prosthetics*. Quintessence: Chicago; 2000; 215-31.
27. Lyons MF, Cameron DA, Banham SW. Snoring , sleep apnea and the role of oral appliances. *Dent Update* 2001; 28:254-256.
28. Ferguson KA. The role of oral appliance therapy in the treatment of obstructive sleep apnea. *Clin Chest Med* 2003; 24:355–364.
29. Cameron DA, Lyons MF, Fox DL, Banham SW. Pilot study of a flexible intra-oral appliance for the control of snoring. *Br Dent J* 1998; 85:304-7.
30. Mohsenin N, Mostofi MT, Mohsenin V. The role of oral appliances in treating obstructive sleep apnea. *J Am Dent Assoc* 2003; 34:442–449.
31. Lindman R, Bondemark L. A review of oral devices in the treatment of habitual snoring and obstructive sleep apnoea. *Swed Dent J*. 2001; 25:39–51.
32. Schoem SR. Oral appliances for the treatment of snoring and obstructive sleep apnea. *Otolaryngol Head Neck Surg*. 2000; 122: 259–262.
33. Bian H. Knowledge, opinions, and clinical experience of general practice dentists toward obstructive sleep apnea and oral appliances. *Sleep Breath* 2000; 8:85–90.
34. Ferguson KA, Love LL, Ryan CF. Effects of a mandibular and tongue protrusion on upper airway size. *Am J Respir Crit Care Med* 1997;155: 1748-54.
35. Jauhar S et al. Ten year follow up of mandibular advancement devices for management of snoring and sleep apnea. *J prosthet Dent* 2008; 99: 314-321
36. McGown AD, Marker H K, Battgel JM, Estrange PR, Grant HR , Spiro SG. Long term use of mandibular advancement splints for snoring and obstructive sleep apnea: a questionnaire survey. *Eur Respir J* 2001;17:462-6.
37. Cartwright RD, Samelson CF. The effects of a nonsurgical treatment for obstructive sleep apnea. The tongue-retaining device. *JAMA* 1982; 248:705–709
38. De Almeida FR, Lowe AA, Tsuiki S, Otsuka R, Wong M, Fastlicht S, Ryan CF. Long term compliance and side effects of oral appliances used for the treatment of snoring and obstructive sleep apnea syndrome. *J Clin Sleep Med* 2005;1:143–152
39. Pantin CG, Hillman DR, Tennant M. Dental side effects of an oral device to treat snoring and obstructive sleep apnea. *Sleep* 1999; 22:237–240